Amendment to the specification:

Replace the original Sequence Listing filed July 15, 2002, with the substitute Sequence Listing filed herewith.

Please amend the paragraph beginning at page 8, line 21, as follows:

Figure 6 is a series of four bar graphs illustrating the cellular responses to class I-restricted peptides from gp120 (class I-restricted peptide from gp120 is depicted in the figure legend as RGPGRAFVTI (SEQ ID NO:[[16]]13)).

Please amend the paragraph beginning at page 8, line 25, as follows:

Figure 8 is a graph plotting the results of a cell lysis assay (JAM). HSVgp120 mediated induction of CTL activity (HIVgp120 specific peptides are depicted in the figure legend as RGPRAFVTI (SEQ ID NO:[[17]]14) and RGPGRAFVTI (SEQ ID NO:[[16]]13)).

Please amend the paragraph beginning at page 8, line 27, as follows:

Figure 9 is a series of four bar graphs illustrating the effect of administering an HSV-gp120 amplicon by three common routes of administration (intramuscular, subcutaneous, or intraperitoneal). The HIVgp120 specific peptide is depicted in the figure legend as RPGRAFVTI (SEQ ID NO:[[18]]15).

Please amend the paragraph beginning at page 20, line 4, as follows:

To determine whether animals immunized with an HSV-gp120 amplicon could later mount a cell-mediated immune response to the gp120 antigen, mice were immunized with either (1) an HSV-gp120 amplicon, (2) a sequence encoding the V3 peptide (MVA.H), or (3) an HSV-lacZ amplicon. "Naïve" mice constituted a fourth group. Following immunization, the mice were sacrificed and their splenocytes were placed in culture. The cellular responses to a class I-restricted peptide from gp120 (RGPGRAFVTI (SEQ ID NO:[[16]] 13)) were measured by interferon gamma Elispot. Splenocytes incubated without the gp120 peptide served as another control for this study. The number of interferon-gamma-positive spots per well was plotted for each animal, in triplicate, with three dilutions of input splenocytes (100,000; 200,000; and

400,000 cells/well). The results are shown in Figure 6. The designations A1-A4 represent splenocytes obtained from individual animals, and the (+) and (-) symbols beneath those designations mark splenocytes incubated with or without the specific gp120 peptide. As shown in Figure 6, the number of interferon gamma-positive spots (which is indicative of the ability of the cells to mount a cell-mediated immune response) was low and not significantly different in splenocytes obtained from mice that were immunized with MVA or HSV-lacZ or that were not immunized at all (naïve). However, significantly more of the splenocytes obtained from HSV-gp120-immunized mice produced interferon following exposure to the gp120 peptide in culture.

Please amend the paragraph beginning at page 21, line 7, as follows:

BALB/c mice (n=3) were inoculated with an HSV-gp120 amplicon (10^6 pfu) by intramuscular injection. The mice were sacrificed 21 days later, and splenocytes were harvested and placed in culture, where they were restimulated in the presence of LPS blasts loaded with the HIVgp120 specific peptide RGPRAFVTI (SEQ ID NO:[[17]]14). After five days, these effector cells were mixed at various ratios with radiolabeled P815 target cells, either pulsed with peptide (+) or unpulsed (-). Cell killing was assessed using the JAM assay method described by Matzinger *et al.* (*J. Immunol. Methods* 145:185-92, 1991). The data, shown in Figure 8, were expressed in terms of % cytotoxicity at each effector to target (E:T) ratio. A1, A2, and A3 denote data obtained from individual animals. These data demonstrate that a single intramuscular injection of an HSV-gp120 vector is sufficient to produce a strong, peptide-specific, cytotoxic effector response in the treated animals.

Please amend the paragraph beginning at page 21, line 22, as follows:

To study the effect of the route of administration on the strength of the immune response generated, BALB/c mice were inoculated with the same vector, an HSV-gp120 amplicon (10^6 pfu) administered either intramuscularly (into the thigh), subcutaneously (at the base of the tail), or intraperiotoneally. Control mice received 10^6 pfu of the HSV-lacZ vector intramuscularly. All animals were sacrificed 21 days later, and their splenocytes were harvested and subjected to an interferon-gamma Elispot assay using either an HIVgp120 specific peptide (RGPRAFVTI (SEQ ID NO:[[17]]14); designated "+" in Figure 9) or no peptide (designated "-" in Figure 9).

A1, A2, and A3 designate splenocytes obtained from individual animals. As shown in Figure 9, while all routes of administration produced some number of interferon-gamma-positive spots per well, the greatest number were produced when the antigen had been administered subcutaneously. Thus, subcutaneous inoculation with HSV-gp120 produced the best cellular immune response (at least as defined in this assay system under the parameters used).

Please amend the paragraph beginning at page 26, line 8, as follows:

TaqMan Quantitative PCR System: To isolate total DNA for quantitation of amplicon genomes in packaged stocks, virions were lysed in 100-mM potassium phosphate pH 7.8 and 0.2% Triton X-100. Two micrograms of genomic carrier DNA was added to each sample. An equal volume of 2X Digestion Buffer (0.2 M NaCl, 20 mM Tris-Cl pH 8.0, 50 mM EDTA, 0.5% SDS, 0.2 mg/ml proteinase K) was added to the lysate and the sample was incubated at 56°C for 4 hrs. Samples were processed further by one phenol:chloroform, one chloroform extraction, and a final ethanol precipitation. Total DNA was quantitated and 50 ng of DNA was analyzed in a PE7700 quantitative PCR reaction using a designed *lacZ*-specific primer/probe combination multiplexed with an 18S rRNA-specific primer/probe set. The *lacZ* probe sequence was 5'-6FAM-ACCCCGTACGTCTTCCCGAGCG-TAMRA-3' (SEQ ID NO:4); the *lacZ* sense primer sequence was 5'- GGGATCTGCCATTGTCAGACAT-3' (SEQ ID NO:5); and the *lacZ* antisense primer sequence was 5'- TGGTGTGGGCCATAATTCAA-3' (SEQ ID NO: [[15]]12). The 18S rRNA probe sequence was 5'-JOE-TGCTGGCACCAGACTTGCCCTC-TAMRA-3' (SEQ ID NO:6); the 18S sense primer sequence was 5'-CGGCTACCACATCCAAGGAA-3' (SEQ ID NO:7); and the 18S antisense primer sequence was 5'-GCTGGAATTACCGCGGCT-3' (SEQ ID NO:8).

Please amend the paragraph beginning at page 34, line 21, as follows:

VP16 is a strong transactivator protein and structural component of the HSV virion (Post *et al.*, *Cell* 24:555-565, 1981). VP16-mediated transcriptional activation occurs via interaction of VP16 and two cellular factors, Oct-1 (O'Hare and Goding, *Cell* 52:435-445, 1988; Preston *et al.*, *Cell* 52:425-434, 1988; Stern *et al.*, *Nature* 341:624-630, 1989) and HCF (wilson *et al.*, *Cell* 74:115-125, 1993; Xiao and Capone, *Mol. Cell Biol.* 10:4974-4977, 1990) and subsequent

binding of the complex to TAATGARAT (SEQ ID NO:16) elements found within HSV IE promoter regions (O'Hare, *Semin. Virol.* 4:145-155, 1993. This interaction results in robust up-regulation of IE gene expression. Neuronal splice-variants of the related Oct-2 transcription factor have been shown to block IE gene activation via binding to TAATGARAT (SEQ ID NO:16) elements (Lillycrop *et al.*, *Neuron* 7:381-390, 1991) suggesting that cellular transcription factors may also play a role in limiting HSV lytic growth.

Please amend the paragraph beginning at page 39, line 12, as follows:

Virus Titering: Helper virus-containing stocks were titered for helper virus by standard plaque assay methods (Geschwind *et al.*, *Brain Res. Mol. Brain Res.* 24:327-335, 1994). Amplicon titers for both helper virus-based and helper-free stocks were determined as follows. NIH 3T3 cells were plated in a 24-well plate at a density of $1x10^5$ cells/well and infected with the virus. Twenty-four hours after viral infection, the monolayers were washed twice in PBS and either fixed with 4% paraformaldehyde and stained by X-gal histochemistry (HSVlac; 5 mM potassium ferricyanide; 5 mM potassium ferrocyanide; 0.02% NP-40; 0.01% sodium deoxycholic acid; 2 mM $MgCl_2$; and 1 mg/ml X-gal dissolved in PBS) or harvested for total DNA using lysis buffer (100 mM NaCl, 10 mM Tris, pH 8.0, 25 mM EDTA, 0.5% SDS) followed by phenol/chloroform extraction and ethanol precipitation. Real-time quantitative PCR was performed on duplicate samples using primers corresponding to the β-lactamase gene present in the amplicon plasmid, according to Bowers *et al.* (*Mol. Ther.* 1:294-299, 2000). Total DNA was quantitated and 50 ng of DNA was analyzed in a PE7700 quantitative PCR reaction using a designed β-lactamase-specific primer/probe combinatino multiplexed with an 18S rRNA-specific primer/probe set. The β-lactamase probe sequence was 5'-CAGGACCACTTCTGCGCTCGGC-3' (SEQ ID NO:9); the β-lactamase sense primer sequence was 5'-CTGGATGGAGGCGGATAAAGT-3' (SEQ ID NO:10); and the β-lactamaseantisense primer sequence was 5'-TGCTGGCACCAGACTTGCCCTC-3' (SEQ ID NO:11). The 18S rRNA probe sequence was 5'-TGCTGGCACCAGACTTGCCCTC-3' (SEQ ID NO:[[12]]6); the 18S sense primer sequence was 5'-CGGCTACCACATCCAAGGAA-3' (SEQ ID NO:[[13]]7); and the 18S antisense primer sequence was 5'-GCTGGAATTACCGCGGCT-3' (SEQ ID NO:[[14]]8). Helper virus titers (pfu/ml), amplicon expression titers (bfu/ml), and amplicon

transduction titers (TU/ml) obtained from these methods were used to calculate amplicontiter and thus standardize experimental viral delivery. Amplicon titers of the various virus preparations ranged from 4-5 x 10^8 bfu/ml while helper titers were in the range of 5-15 x 10^7 pfu/ml.